RESEARCH PAPER

SPECTRUM OF OCULAR SURFACE DISORDERS IN TYPE 2 DIABETES MELLITUS

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ABSTRACT

Aim

This study aimed to assess association between diabetes and ocular surface diseases and to evaluate impact of disease on visual function and quality of life, and establish a screening and management protocol for ocular surface diseases.

Materials and Methods

This observational, cross-sectional study enrolled a total of 148 participants, including 74 with T2DM and 74 age and sex matched healthy controls. The study assessed several ocular surface parameters, including tear-film break-up time, Schirmer test, corneal sensitivity, meibomian gland dysfunction, and the Ocular Surface Disease Index score. Data collection occurred for period of 8 months, and statistical analysis was conducted using multivariable logistic regression and linear regression models to determine relationship between diabetes-related factors and ocular surface disease severity.

Results

The study found significantly higher prevalence of ocular surface disorders (OSD) in diabetics compared to controls. Dry-eye disease affected 59.5% of diabetics, with meibomian gland dysfunction in 51.4% and superficial punctate keratopathy in 28.4%. Diabetics had notably reduced tear-film stability and production. Poor metabolic control, longer disease duration, and diabetic retinopathy were identified as independent risk factors for OSD. HbA1c levels, particularly ≥ 9%, were strongly correlated with the severity of ocular surface damage.

Conclusion

Diabetes mellitus, particularly with poor glycaemic control and long duration, significantly contributes to the development of ocular surface disorders. The findings emphasize need for regular ocular screening in diabetics, particularly those with poor metabolic control. Developing a targeted screening and management protocol for ocular surface diseases in this population to improve visual function and quality of life.

Keywords: Diabetes mellitus, Ocular surface disorders, Dry eye disease, Meibomian gland dysfunction, Diabetic retinopathy, HbA1c.

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INTRODUCTION

Diabetes mellitus is a metabolic disorder marked by chronic hyperglycaemia, causing systemic and ocular complications, including ocular surface disease. The ocular surface—comprising the cornea, conjunctiva, tear film, and associated neural and vascular components—maintains visual clarity and comfort. In type 2 diabetes (T2DM), prolonged hyperglycaemia disrupts tear-film homeostasis, leading to decreased aqueous secretion from lacrimal glands, lipid-layer insufficiency from meibomian-gland dysfunction, and mucin deficiency from goblet-cell loss. These changes increase tear osmolarity, triggering inflammation, epithelial damage, and dry eye disease [1,2].

The cornea is highly vulnerable: metabolic disturbances, oxidative stress, and inflammatory mediators impair epithelial function, delay wound healing, and increase infection risk. Diabetic

neuropathy reduces corneal sensitivity, predisposing to neurotrophic keratopathy [3]. Chronic hyperglycaemia promotes advanced glycation end-product formation, activating inflammatory pathways that destabilize the epithelium [4]. Microvascular dysfunction further compromises oxygen and nutrient delivery, while elevated VEGF contributes to vascular changes and delayed healing [5].

Meibomian-gland dysfunction, oxidative stress, and reduced neurotrophic factors exacerbate tear-film instability and epithelial fragility [6–9]. Poor glycaemic control correlates with greater ocular surface damage, emphasizing the need for early diagnosis, metabolic optimization, and targeted interventions to prevent progression and preserve ocular health in patients with T2DM [10].

MATERIALS AND METHODS

Study design

The study was a hospital-based, observational, cross-sectional investigation comparing ocular surface status in adults with T2DM and age- and sex-matched nondiabetic controls. Primarily descriptive, it also analysed associations between tear-film parameters and glycaemic variables. Conducted in a tertiary-care ophthalmology outpatient department over eight months, all assessments occurred during a single visit to avoid intervention effects. Examination rooms maintained controlled conditions (45% ± 5% humidity, 24°C ± 2°C) to minimize variability in TBUT and Schirmer measurements.

Inclusion Criteria

- Age ≥ 40 years
- Documented diagnosis of type 2 diabetes mellitus for at least one year based on ADA 2024 criteria
- Ability and willingness to provide written informed consent

Exclusion Criteria

- Use of topical ocular medications.
- History of ocular surgery, trauma, or contact-lens wear within the previous 3 months
- Presence of pterygium, active ocular infection or thyroid-associated orbitopathy .
- Systemic conditions known to affect the ocular surface (e.g- rheumatoid arthritis, Sjögren syndrome)
- Pregnancy or lactation.
- Non cooperative patients.

Sampling and Sample size

A systematic sampling approach enrolled every second eligible T2DM patient and every third non-diabetic attendant, ensuring temporal dispersion, practicality, and reasonable randomness in the high-volume outpatient setting.

Sample size was calculated using the single population proportion formula:

$$n = Z^2P(1-P)/d^2$$

where **Z = 1.96** for 95% confidence, **P = 50%** anticipated prevalence of ocular surface disease in T2DM (chosen to maximise variance), and **d = 12%** absolute precision. This calculation yielded a minimum sample size of approximately 67 participants per group. Allowing for a 10% non-evaluable rate due to incomplete examinations or data loss, the final sample size was rounded to **74 diabetic patients and 74 controls**, giving a total analytic sample of **148 eyes per group**, with the **right eye prespecified for primary analysis**.

Participants were divided into two primary groups:

- **Group A (T2DM):** Adults diagnosed with type-2 diabetes mellitus meeting inclusion criteria
- **Group B (Controls):** Nondiabetic adults matched for age (±3 years) and sex without systemic or ocular conditions listed in the exclusion criteria

Within the diabetic group, secondary subgrouping was performed based on glycated haemoglobin (HbA1c):

- Good glycaemic control: HbA1c <7%
- Moderate control: HbA1c 7–8.9%
- Poor control: HbA1c ≥9%

This classification enabled evaluation of dose-response relationships between glycaemic control and ocular surface parameters.

The **primary outcome** was the prevalence of ocular surface disorder, defined as the presence of at least one abnormal clinical sign:

- TBUT <10 seconds
- Schirmer I ≤5 mm/5 min
- Fluorescein staining grade ≥1
- Rose Bengal staining score ≥3
- Meibomian gland dysfunction ≥ Grade 2

Secondary outcomes included:

- TBUT
- Schirmer I and II test values
- Corneal sensitivity threshold
- Meibomian gland dysfunction grading
- Ocular Surface Disease Index score
- Best-corrected visual acuity (LogMAR)
- Pelli-Robson contrast sensitivity
- Glycaemic parameters (fasting glucose, postprandial glucose, HbA1c)
- Duration of diabetes and stage of diabetic retinopathy (ETDRS classification)

Clinical Examination Procedure

To minimise reflex tearing and measurement bias, tests were performed in a standardised sequence. Ambient environmental parameters were recorded first. Best-corrected visual acuity was assessed using an illuminated Snellen chart at 6 m. Objective refraction was obtained using autorefractor and refined subjectively. Contrast sensitivity was

measured monocularly using the Pelli-Robson chart at 1 m under photopic illumination.

TBUT was assessed using a fluorescein strip moistened with non-preserved saline and applied to the inferior conjunctival sac without topical anaesthesia. Participants were asked to blink three times and keep their eyes open. The interval between the final blink and the appearance of the first dry spot in the precorneal tear film was measured. Three readings were taken and the mean value recorded.

After a five-minute interval, **Schirmer I testing** was performed without anaesthesia using Whatman filter paper strips placed at the junction of the middle and lateral third of the lower eyelid. Wetting length was measured after five minutes. Subsequently, topical proxymetacaine 0.5% was instilled, and **Schirmer II testing** was conducted to evaluate basal tear secretion.

Corneal sensitivity was assessed using the cotton-swab method.

Fluorescein and Rose Bengal staining patterns were graded using the modified Oxford grading scheme and the van Bijsterveld scoring system respectively.

Systemic biochemical parameters, including fasting plasma glucose, postprandial glucose, and HbA1c (measured using HPLC on a Bio-Rad D-10 analyser), were obtained on the same day.

For diabetic participants, dilated fundus photography was performed using a fundus camera and graded for diabetic retinopathy according to the ETDRS classification within one week of ocular surface assessment.

All instruments were recalibrated weekly. To maintain consistency, a single examiner conducted all tear-film tests, while a masked senior consultant reviewed a random 10% of recorded examinations to ensure grading reliability.

Statistical analysis was performed using **R software version 4.3.1**. Continuous variables were summarised as mean ± standard deviation for normally distributed data or median with interquartile range otherwise. Group comparisons were conducted using independent-samples t-tests or Mann–Whitney U tests, while categorical variables were analysed using chi-square or Fisher's exact tests.

Multivariable logistic regression was used to identify independent predictors of ocular surface disorders, including age, sex, duration of diabetes, HbA1c level, presence of diabetic retinopathy, and peripheral neuropathy. Model calibration was assessed using the Hosmer–Lemeshow test and discrimination using receiver operating characteristic (ROC) analysis.

For continuous ocular surface parameters such as TBUT and Schirmer values, linear regression with robust standard errors was used to estimate the effect of HbA1c changes. A two-tailed p-value <0.05 was considered statistically significant. Missing data (<3%) were handled using stochastic regression imputation.

RESULTS

The demographic characteristics of the two groups were comparable, with no significant differences in age (p=0.58) or sex distribution (p=0.74). However, body mass index was significantly higher in the diabetic group compared to controls (27.3±3.9 vs 25.8±4.1 kg/m², p=0.02). Smoking prevalence was similar between groups (12.2% vs 10.8%, p=0.80) **[Table-1].**

Systemic comorbidities were significantly more frequent among diabetic participants. Hypertension was present in 55.4% of diabetics compared to 21.6% of controls (p<0.001), while dyslipidaemia was observed in 51.4% versus 17.6% respectively (p<0.001). Peripheral neuropathy, assessed by monofilament testing, was detected only in diabetics (32.4% vs 0%, p<0.001).

Visual function parameters also differed significantly between groups. Best-corrected visual acuity was worse in diabetics (LogMAR 0.18±0.21 vs 0.06±0.09, p<0.001), and contrast sensitivity was reduced (1.45±0.28 vs 1.68±0.19, p<0.001). Lens opacity was more common among diabetics (37.8% vs 16.2%, p=0.003), while intraocular pressure remained comparable (p=0.18). **[Figure-1]**

Among diabetic participants , 75.7% showed some degree of diabetic retinopathy. Mild non-proliferative diabetic retinopathy (NPDR) was the most frequent (n=24, 32.4%), followed by moderate NPDR (n=20, 27%), while severe NPDR or proliferative retinopathy was observed in (n=12 ,16.2%) of patients. **[Figure-2]**

Ocular surface disorders (OSD) were significantly more prevalent in diabetics **[Table-2].** Overall OSD prevalence was 63.5% among diabetics compared to 22.9% in controls (p<0.001). Dry eye disease was present in 59.5% of diabetics versus 20.3% of controls. Meibomian gland dysfunction (grade ≥2) occurred in 51.4% of diabetics compared to 17.6% of controls, and superficial punctate keratopathy was also more frequent (28.4% vs 5.4%). Reduced corneal sensitivity all showed significant risk elevation.

Tear film parameters deteriorated with increasing diabetes duration **[Table-3].** Tear break-up time decreased from 8.1±1.8 seconds in patients with ≤5 years of diabetes to 4.9±1.3 seconds in those with >10 years (p<0.001). Schirmer I test values showed a similar decline (9.4±3.6 mm vs 5.2±2.8 mm).

Meibomian gland dysfunction severity **[Table-4]** was significantly greater in diabetics. Normal clear secretion (grade 0) was present in only 16.2% of diabetics versus 43.2% of controls. Grade 2 dysfunction with turbid secretion affected 35.1% of diabetics versus 13.5% of controls. Complete absence of secretion (grade 3) was four times more common in diabetics (16.2% vs 4.1%).

Corneal sensitivity decreased with worsening glycaemic control **[Figure-3],** with patients having HbA1c ≥9% demonstrating significantly reduced sensitivity compared to those with HbA1c <7% ($p<0.001$).

OSD frequency paralleled retinopathy severity: from 39 % in eyes without DR to 92 % in those with severe NPDR/PDR. The shared microvascular aetiology implies that patients needing retinal follow-up also warrant anterior-segment evaluation, enabling an integrated approach to diabetic eye disease **[Table-5].**

Subjective symptom burden paralleled objective findings: diabetics reported a median OSDI of 25, double that of controls. Although neurotrophic loss can mask discomfort, the significant difference ($P< 0.001$) indicates that many diabetic patients do perceive bothersome dryness and visual fluctuation, validating the clinical relevance of screening questionnaires alongside slit-lamp signs. **[Figure-4].**

OSD prevalence increased progressively with poorer glycaemic control and greater retinopathy severity. Multivariable logistic regression **[Table-6]** identified diabetes duration (OR 1.18), HbA1c (OR 1.52), diabetic retinopathy (OR 2.34), and peripheral neuropathy (OR 2.76) as independent predictors of OSD.

Linear regression analysis demonstrated that TBUT fell by 0.78 s for every 1 % rise in HbA1c and by 0.18 s per diabetes year. Higher MGD grade exerted an independent negative effect. With $R^2 = 0.56$, the model explains over half of TBUT variance and supports combined metabolic and lid-margin interventions to prolong tear stability **[Table-7].**

Receiver-operating characteristics yielded an AUC of 0.80, indicating good prognostic utility of HbA1c. A threshold of 8.5 % balanced sensitivity (68 %) and high specificity (92 %), offering a practical cut-point beyond which targeted ocular-surface screening is especially warranted.

DISCUSSION

The primary aim of this study was to quantify and characterize the spectrum of ocular surface disorders (OSD) in patients with type 2 diabetes mellitus from an Indian tertiary-care population and to examine their relationship with three systemic factors: glycaemic control (HbA1c), duration of diabetes, and stage of diabetic retinopathy. By integrating subjective symptom scores with objective assessments of tear-film stability, aqueous secretion, meibomian gland function, and corneal sensitivity, the study sought to estimate prevalence and determine whether ocular surface damage represents a coincidental finding or a true metabolic complication of diabetes.

This investigation addresses an important evidence gap. While diabetic retinopathy screening is routinely performed, anterior segment complications remain under-recognised, particularly in South Asian populations where environmental, dietary, and genetic factors may influence tear dynamics. The findings also have direct clinical relevance: approximately two-thirds of adults with diabetes were found to harbour treatable tear-film or lid-gland pathology, and clear systemic risk thresholds were identified—an HbA1c level around 8.5% and a disease duration exceeding ten years. These thresholds provide practical triggers for clinicians to initiate early ocular surface evaluation, preventive lid hygiene, and lubricating therapy. Furthermore, the strong association between ocular-surface parameters and systemic metabolic burden suggests that these ocular findings may serve as surrogate markers of microvascular health.

Using a composite diagnostic endpoint—TBUT <10 seconds, Schirmer ≤10 mm, fluorescein or Rose-Bengal staining ≥ grade 1, meibomian gland dysfunction (MGD) ≥ grade 2, or reduced corneal sensitivity—the prevalence of ocular surface disorders was 63.5% among diabetic participants compared with 22.9% in controls (risk ratio 2.78, 95% CI 1.78–4.34, $P < 0.001$). These findings align with previous studies. Naik et al.[11] reported a 67% prevalence of dry eye in patients with HbA1c >8%, while Xin et al.[12] documented symptomatic dry eye in 70.8% of diabetics versus 27.5% of controls. Liang et al.[13] observed objective signs in 55% of diabetic patients. Variations between studies likely reflect differences in diagnostic criteria, with questionnaire-based approaches tending to yield higher prevalence rates. Our broader diagnostic definition captured both evaporative and neurotrophic mechanisms underlying ocular surface disease.

Specific ocular surface abnormalities were also more frequent in diabetic patients. Superficial punctate keratopathy occurred in 28.4% of diabetics—five times the prevalence seen in controls—similar to the 30% rate reported by Gao et al.[14] in proliferative diabetic retinopathy cohorts. Reduced corneal sensitivity was detected in 39.2% of diabetics, exceeding the 23% reported by DeMill et al.[15], possibly due to stricter aesthesiometry thresholds in our protocol. These findings underscore the role of epithelial fragility and neurotrophic impairment in diabetic ocular surface disease.

Disease duration demonstrated a strong dose–response relationship with tear-film parameters. Tear break-up time declined from 8.1 ± 1.8 seconds in patients with diabetes duration ≤5 years to 4.9 ± 1.3 seconds in those with disease longer than ten years. Schirmer values similarly decreased from 9.4 mm to 5.2 mm. These patterns closely mirror findings by He et al., who reported a similar decline

in non-invasive break-up time across comparable duration groups. Correlation analysis in our study showed significant inverse relationships between duration and both TBUT (ρ = –0.58) and Schirmer values (ρ = –0.46). These changes likely reflect cumulative metabolic injury, including sorbitol pathway activation, lacrimal gland microangiopathy, and inflammatory cytokine activity such as TNF-α. Clinically, these results suggest that tear-film parameters could function as indicators of cumulative glycaemic burden, analogous to microalbuminuria in diabetic nephropathy.

Meibomian gland dysfunction emerged as another major contributor to ocular surface disease. Moderate or severe MGD affected 51.4% of diabetic patients compared with 17.6% of controls (risk ratio 2.92). Grade 3 MGD, indicating absent gland secretion, was observed in 16.2% of diabetics, four times higher than in controls. These results are consistent with Liang et al., who reported similar rates of meibomian gland loss using infrared meibography. Hyperglycaemia and hyperinsulinemia may impair androgen-regulated lipid synthesis in glandular acinar cells, leading to evaporative tear-film instability. Clinically, these findings support routine lid-margin examination and consideration of therapies such as warm compresses, lid hygiene, omega-3 supplementation, and lipid-based lubricants during diabetic eye evaluations.

Corneal sensitivity also showed a clear relationship with glycaemic control. Patients with HbA1c levels ≥9% required significantly shorter filament lengths on aesthesiometry (43 ± 5 mm) compared with well-controlled individuals with HbA1c <7% (51 ± 4 mm). These results parallel observations by Gao et al.[15] and Han et al.[16], who reported progressive corneal nerve fibre loss with worsening diabetic retinopathy. Neurotrophic compromise may therefore occur silently, as symptoms often increase less dramatically than objective sensory deficits. Reduced corneal sensation also carries important clinical implications, as diminished blink reflex and impaired epithelial healing increase the risk of corneal ulceration.

Subjective symptom burden, measured using the Ocular Surface Disease Index (OSDI), was significantly greater among diabetics, with median scores nearly doubling compared with controls (25 vs 10). Moderate inverse correlations were observed between OSDI and both TBUT (ρ = –0.56) and Schirmer values (ρ = –0.50). However, as previously described by DeMill et al.[15], symptoms and signs may diverge once neurotrophic damage becomes advanced. This finding highlights the importance of combining symptom questionnaires with objective testing during screening.

Glycaemic control showed a strong association with OSD prevalence. The proportion of affected individuals increased from 42.1% among patients with HbA1c <7% to 81% in those with HbA1c ≥9%. Logistic regression confirmed HbA1c as an independent predictor of ocular surface disease, with an adjusted odds ratio of approximately 2.03 per percentage increase. These findings support mechanistic hypotheses that chronic hyperglycaemia leads to advanced glycation end-product deposition, epithelial dysfunction, and impaired wound healing.

Further analysis demonstrated that tear-film stability was influenced by multiple factors. Linear regression showed that TBUT shortened by 0.78 seconds for each 1% increase in HbA1c, by 0.18 seconds per additional year of diabetes duration, and by 0.92 seconds with each increase in MGD grade. These results highlight the additive impact of metabolic and glandular dysfunction on tear-film stability and emphasize the therapeutic potential of lid-margin interventions.

Receiver-operating-characteristic analysis demonstrated that HbA1c had moderate diagnostic utility for predicting ocular surface disease (AUC 0.80). An HbA1c threshold of approximately 8.5% yielded 68% sensitivity and 92% specificity, suggesting that this parameter could function as a practical triage marker in resource-limited settings. Electronic medical record alerts based on such thresholds may facilitate timely referral for ocular surface evaluation.

Correlation analysis further demonstrated strong relationships among tear metrics. TBUT correlated positively with Schirmer values and inversely with MGD grade and OSDI score, indicating that aqueous deficiency, lipid layer insufficiency, and evaporative instability interact to produce ocular surface symptoms.

Several methodological strengths enhance the credibility of this study. Rigorous age- and sex-matching minimized demographic confounding, while the inclusion of a well-matched control group strengthened comparative analysis. A comprehensive battery of both subjective and objective diagnostic tools ensured a multifactorial evaluation of ocular surface health. Testing was conducted in a standardized sequence under controlled environmental conditions to reduce physiologic variability. Stratification by glycaemic control, disease duration, and retinopathy stage allowed detailed dose–response analysis and supported multivariable modelling. Inter-observer reliability exceeded accepted benchmarks, confirming measurement consistency, and adverse events during testing were minimal.

Limitations

Several limitations merit consideration. The cross-sectional design precludes causal inference, and longitudinal studies are needed to establish temporal relationships. Single-center recruitment may limit generalizability, particularly to different climatic conditions affecting tear film stability. The

exclusion of patients using artificial tears may underestimate true OSD prevalence in the diabetic population. Additionally, the study did not assess inflammatory markers or tear osmolarity, which could provide mechanistic insights.

CONCLUSION

This study demonstrates that ocular surface disorders are highly prevalent among individuals with type 2 diabetes and are closely linked to systemic metabolic status. Poor glycaemic control, longer disease duration, and diabetic retinopathy significantly increase the risk of tear-film dysfunction, meibomian gland disease, and corneal neurotrophic impairment. These findings support the integration of ocular surface assessment into routine diabetic eye care, thereby promoting earlier detection, targeted management, and a more comprehensive approach to preserving visual health in patients with diabetes

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[Table-1]: Demographic Profile of Study Groups

Parameter	Group A (n=74)	Group B (n=74)	p-value
Age (years)	58.6±9.3	57.9±8.8	0.58
Female, n (%)	31 (41.9)	29 (39.2)	0.74
BMI (kg/m²)	27.3±3.9	25.8±4.1	0.02
Current smoker, n (%)	9 (12.2)	8 (10.8)	0.80

[Table-2]: Prevalence of Ocular Surface Disorders

Disorder	Group A (n=74)	Group B (n=74)	Risk Ratio	p-value
Any OSD	47 (63.5%)	17 (22.9%)	2.78	<0.001
Dry eye disease	44 (59.5%)	15 (20.3%)	2.93	<0.001
MGD grade ≥2	38 (51.4%)	13 (17.6%)	2.92	<0.001
SPK	21 (28.4%)	4 (5.4%)	5.26	<0.001
Reduced corneal sensitivity	29 (39.2%)	6 (8.1%)	4.83	<0.001

[Table-3]: Tear Film Metrics by Diabetes Duration

Duration	n	TBUT (s)	Schirmer I (mm)
≤5 years	21	8.1±1.8	9.4±3.6
6-10 years	27	6.2±1.7	7.0±3.2
>10 years	26	4.9±1.3	5.2±2.8

[Table-4]: Meibomian Gland Dysfunction Grading

Grade	Definition	Group A n (%)	Group B n (%)
0	Clear fluid	12 (16.2)	32 (43.2)
1	Plugging, serous secretion	24 (32.4)	29 (39.2)
2	Plugging, turbid secretion	26 (35.1)	10 (13.5)
3	No secretion	12 (16.2)	3 (4.1)

[Table-5]: Retinopathy Stage and OSD Risk

DR Stage	n	OSD n (%)
None	18	7 (38.9)
Mild NPDR	24	14 (58.3)
Moderate NPDR	20	15 (75.0)
Severe NPDR/PDR	12	11 (91.7)

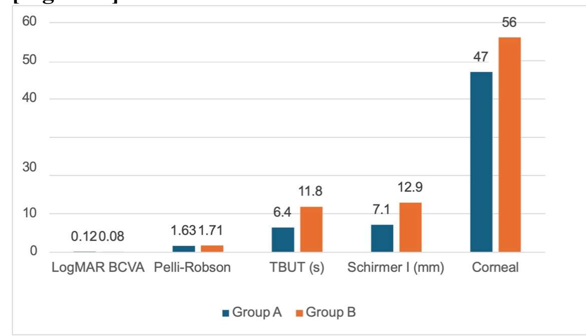
[Table-6]: Predictors of OSD (Multivariable Model)

Variable	Adjusted OR	95% CI	p-value
Diabetes duration (per year)	1.18	1.09-1.28	<0.001[HS]
HbA1c (per 1% increase)	1.52	1.21-1.91	<0.001[HS]
Presence of DR	2.34	1.12-4.89	0.024[S]
Peripheral neuropathy	2.76	1.31-5.82	0.008[S]
Age (per year)	1.02	0.98-1.06	0.35[NS]
Female sex	1.14	0.56-2.32	0.72[NS]

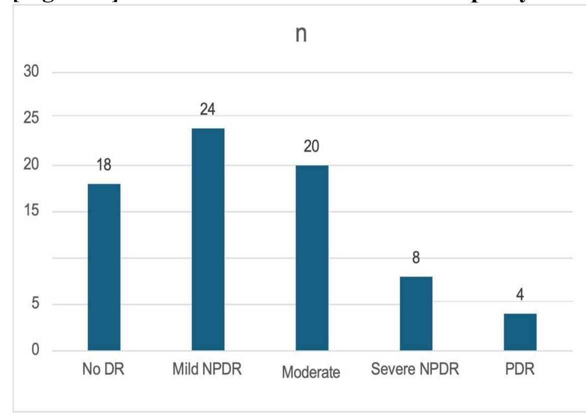
[Table-7]: Determinants of TBUT (Linear Regression)

Variable	β-coefficient	95% CI	p-value
HbA1c (per 1%)	-0.84	-1.12 to -0.56	<0.001
Duration (per year)	-0.31	-0.42 to -0.20	<0.001
MGD grade	-1.23	-1.68 to -0.78	<0.001

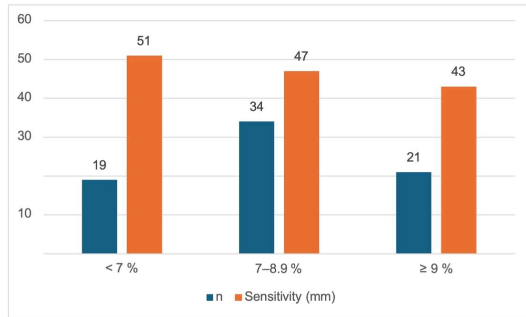
[Figure-1] - Baseline Ocular Parameters



[Figure-2] - Distribution of Diabetic Retinopathy



[Figure-3] - Corneal Sensitivity by Glycaemic Control



[Figure-4] - Ocular Surface Disease Index Scores